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Nerolidol Protects Against LPS-induced Acute Kidney Injury via Inhibiting TLR4/NF-κB Signaling

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Abstract

Acute kidney injury (AKI) is a critical care syndrome, resulting in acute reduction of renal function and up to 22% mortality of hospitalized patients. Nerolidol is a major component in several essential oils that possesses various pharmacological properties. The present study aimed to investigate the potential effect of nerolidol on lipopolysaccharide (LPS)-induced AKI. Nerolidol dose-dependently reduced the pathological injuries of kidney induced by LPS in rats. Nerolidol significantly decreased the levels of blood urea nitrogen and creatinine in LPS-treated rats in a dose-dependent manner. In addition, nerolidol inhibited LPS-induced decrease of cell viability in NRK-52E rat proximal tubular cells, which effect was concentration dependent. Nerolidol notably inhibited the increase of TNFα and IL-1β in LPS-treated rats and the mRNA expression of TNFα and IL-1β in LPS-treated NRK-52E cells. Nerolidol suppressed the increase of toll-like receptor 4 (TLR4) expression, phosphorylation and nuclear translocation of p65 NF-κB in kidneys of LPS-treated rats and LPS-treated NRK-52E cells. Overexpression of TLR4 and p65 NF-κB significantly suppressed nerolidol-induced inhibition of TNFα and IL-1β expression and increase of cell viability in LPS-treated cells. In summary, we found that nerolidol played a critical anti-inflammatory effects through inhibition of TLR4/NF-κB signaling and protected against LPS-induced AKI. Copyright © 2017 John Wiley & Sons, Ltd.

Keywords: NF-κB; acute kidney injury; lipopolysaccharide; nerolidol; toll-like receptor 4.

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